Furan: Human health tier II assessment

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CAS Number: 110-00-9

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

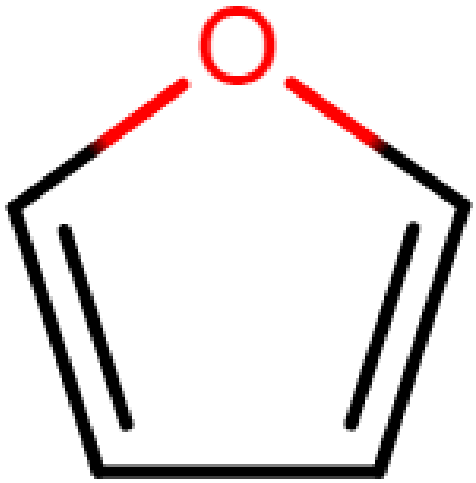
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:www.nicnas.gov.au

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	1,3-epoxy-1,3-butadiene oxacyclopentadiene furfuran divinylene oxide
Structural Formula	
Molecular Formula	C4H4O
Molecular Weight (g/mol)	68.07
Appearance and Odour (where available)	Clear colourless liquid with a strong ether-like odour
SMILES	C1C=COC=1

Import, Manufacture and Use

Australian

No specific Australian use, import, or manufacturing information has been identified.

International

The following international uses have been identified through: the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments (National Toxicology Program (NTP)).

The chemical has reported site-limited uses, including:

- as an intermediate for manufacturing other chemicals (tetrahydrofuran, pyrrole, and thiophene);
- as a solvent for resins; and
- in the production of stabilisers and lacquers.

The chemical has reported non-industrial uses, including in the production of agricultural chemicals and pharmaceuticals.

Restrictions

Australian

No known restrictions have been identified.

International

The chemical is listed on the following (Galleria Chemica):

- EU Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain; and
- The Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex II Part 1: List of substances which must not form part of the composition of cosmetic products.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

- Xn; R20/22 (acute toxicity);

- Xn; R48/22 (repeat dose toxicity);
- Xi; R38 (irritation);
- R45 Carc. Cat 2 (carcinogenicity); and
- R68 Mut. Cat 3 (mutagenicity).

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standards are identified (Galleria Chemica).

Time weighted average (TWA) of:

- 0.5 (mg/m^3) in Bulgaria, China and Latvia; and
- 0.5–1.5 (mg/m^3) in Russia.

Health Hazard Information

Furan is found naturally in very small concentrations in a range of substance including some foods (FSANZ, 2004).

Toxicokinetics

The chemical is highly volatile and can be absorbed following oral, dermal and inhalation exposure. The low polarity of the chemical enables diffusion through biological membranes. Following oral gavage dosing at 8 mg/kg bw in male Fischer 344 (F344) rats, the chemical was rapidly absorbed into the lungs or intestines. It was found mainly in the liver and kidneys for 24 hours. It was found to bind to liver protein but not with liver DNA. About 20 % of the administered dose was excreted in the urine, 22 % in the faeces and 26 % as carbon dioxide (IARC, 1995; NTP, 1996).

The chemical was extensively metabolised following oral ingestion. In male rats, 14 % of the administered dose was excreted unchanged. The primary reactive and cytotoxic metabolite of the chemical was identified as *cis*-2-butene-1,4-dial (BDA), which irreversibly binds to proteins and nucleosides forming adducts. Oxidation of the chemical to this metabolite is catalysed by cytochrome P450 enzymes. The reaction of BDA with cellular nucleophiles such as glutathione (GSH) and protein form other less reactive metabolites found in urine (IARC, 1995; NTP, 1996; EFSA, 2004; HSDB).

Systemic availability of the chemical is limited by the relatively high retention of the chemical in the liver. The chemical is postulated to undergo extensive first-pass metabolism in the liver (NTP, 1996; EFSA, 2004).

Following inhalation of the vapours, about 90–95 % of the chemical was absorbed in dogs. The amount of the chemical retained in the respiratory tract was proportional to the concentration of the chemical in inhaled vapours (NTP, 1996).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase ‘Harmful if swallowed’ (Xn; R22) in the Hazardous Substances Information System (HSIS) (Safe Work Australia). Only qualitative data are available and there are no LD50 data to support or amend this classification.

There are no available acute oral toxicity studies conducted with the chemical reporting the median lethal dose in animals. However, there are some animal data indicating the chemical to have moderate acute oral toxicity.

Following oral administration (dose not reported), the chemical has a corrosive effect on the mucuous membranes of the mouth in animals. A copious flow of bloody saliva and watery fluid from the nose was observed (HSDB).

A single oral administration of the chemical at 250 mg/kg bw caused evident liver DNA damage in a comet assay in mice (see **Genotoxicity**) (HSDB).

In short-term (16–28 days) repeated dose oral toxicity studies, mortalities have been reported at $\geq$80 mg/kg bw/day in rats and at 160 mg/kg bw/day in mice (IARC, 1995; VKM, 2012; REACH) (see **Repeat dose toxicity**).

Dermal

No data are available.

Inhalation

The chemical is classified as hazardous with the risk phrase ‘Harmful by inhalation’ (Xn; R20) in the HSIS (Safe Work Australia). The LC50 for rats calculated below supports this classification.

The one hour median lethal concentration (LC50) is 3398 ppm (equivalent to a 4-hour LC50 of 4.7 mg/L, calculated according to GHS criteria) in rats and 120 mg/m^3 in mice. Reported signs of toxicity include respiratory distress, decreased blood pressure and convulsions in rats, and hyperactivity followed by laboured breathing in mice (HSDB, REACH).

Corrosion / Irritation

Skin Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to skin' (Xi; R38) in the Hazardous Substances Information System (HSIS) (Safe Work Australia). The limited data available support this classification.

The chemical was reported to be irritating and allergenic when applied at 1–50 % concentrations in solutions to the skin of guinea pigs (HSDB). No further details are available.

Eye Irritation

No animal data are available.

Observation in humans

The chemical is reported to irritate and burn the skin and eyes in humans following short term exposure. The vapours irritate the respiratory tract (HSDB). However, this information is not sufficient to classify the chemical as an eye irritant or a respiratory

irritant.

Sensitisation

Skin Sensitisation

Only limited data are available.

The chemical was reported to be allergenic when applied on the skin of guinea pigs at 1–50 % concentrations (HSDB). Study details are not available.

Repeated Dose Toxicity

Oral

The chemical is classified as hazardous with the risk phrase 'Harmful: Danger of serious damage to health by prolonged exposure if swallowed' (R48/22) in HSIS (Safe Work Australia). The data available support this classification.

Animal studies show that the liver is the primary target organ for the toxicity of the chemical, with rats being more sensitive than mice. Liver toxicity with biliary tract hyperplasia and cholangiofibrosis was observed at 4 mg/kg bw/day in rats in 13-week oral toxicity studies, although these lesions were considered as 'minimally severe'. Kidney effects and atrophy of the testes and ovary were observed at ≥30 mg/kg bw/day (NTP, 1993).

In repeated dose toxicity studies conducted by the National Toxicology Program (NTP), F344 rats (male and female) and female mice (n = 10/sex/dose) were administered (by gavage) the chemical at doses of 0, 4, 8, 15, 30 or 60 mg/kg bw/day for 13 weeks. Male mice were administered the chemical at doses of 0, 2, 4, 8, 15 or 30 mg/kg bw/day. In rats, effects observed included dose dependent statistically significant increases in liver lesions (biliary tract hyperplasia and cholangiofibrosis) at all doses, kidney lesions (tubule dilatation and tubule epithelium necrosis) at ≥30 mg/kg bw/day, increased absolute and relative liver and kidney weights at ≥15 mg/kg bw/day, decreased thymus weights at ≥30 mg/kg bw/day, and atrophy of the thymus, testes, or ovaries at 60 mg/kg bw/day. In mice, effects observed included increased incidences of liver lesions (biliary tract hyperplasia and cholangiofibrosis) in females at ≥30 mg/kg bw/day, and increased incidences of hepatocyte cytomegaly in males at 30 mg/kg bw/day and in females at ≥30 mg/kg bw/day, and increased absolute and relative liver weights at ≥15 mg/kg bw/day. Minimal liver lesions were observed in one male mouse at 8 mg/kg bw/day, and in three female mice at 15 mg/kg bw/day. A no observed adverse effect level (NOAEL) of 2 mg/kg bw/day was established (NTP, 1993; EFSA, 2004; HSDB).

In a 90-day repeated oral toxicity study in F344 rats, the chemical was administered at an extended dose range compared to the NTP study, at 0, 0.03, 0.12, 0.5, 2.0, or 8.0 mg/kg bw/day. Changes in clinical chemistry parameters related to thyroid function were identified by dose-related increases of serum thyroxine (T4) and triidothyronine (T3) in males at ≥0.12 mg/kg bw/day and in females at ≥2 mg/kg bw/day. Significant reductions in serum liver enzymes were observed in all animals at ≥2 mg/kg bw/day. Mild histological lesions of the liver (apoptotic hepatocytes, Kupffer cells with yellow pigment and inflammatory infiltrate) were observed at ≥0.12 mg/kg bw/day. Biliary tract hyperplasia and cholangiofibrosis occurred in males with increased incidences at ≥2 mg/kg bw/day, and in females at the highest dose. These lesions were related to the development of cholangiocarcinomas in rats, as reported by the NTP previously. No adverse effects on testosterone synthesis were observed, despite altered intratesticular testosterone levels. A NOAEL of 0.03 mg/kg bw was derived for hepatic toxicity (VKM, 2012; HSDB). Although hyperplasia and cholangiofibrosis were observed at a lower dose, these lesions are not considered significantly severe and are not sufficient to recommend an amendment for a higher hazard classification.

In a 90-day study, a NOAEL of 0.12 mg/kg bw was established in mice based on hepatic toxicity at 0.5 mg/kg bw/day. Serum alanine aminotransferase (ALT) levels were elevated at 2 mg/kg bw/day, and were increased three-fold at the highest dose of 8 mg/kg bw/day (Gill et al., 2011).

Several short term (16–28 days) repeated dose oral toxicity studies were conducted in mice and rats. Mortalities occurred at 160 mg/kg bw/day in mice and at ≥80 mg/kg bw/day in rats. Increased hepatocyte proliferation and liver necrosis associated to

regenerative hyperplasia were observed in mice at 8 mg/kg bw/day. Rapid development of severe cholangiofibrosis and severe hepatocellular necrosis were observed in rats at ≥45 mg/kg bw/day (IARC, 1995; VKM, 2012; HSDB; REACH).

In a two-year study, liver effects (biliary tract fibrosis, hyperplasia, chronic inflammation, proliferation and hepatocyte cytomegaly, cytoplasmic vacuolisation, degeneration, and necrosis) were observed in both rats and mice at all doses, after six weeks of treatment (2, 4, or 8 mg/kg bw in rats and 8 or 15 mg/kg bw in mice) (NTP, 1993).

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

Short-term exposure to the vapour of the chemical is reported to cause pulmonary oedema at high concentrations. The vapours are also CNS depressants and can cause neurological symptoms including headache, dizziness, shortness of breath, fatigue, unconsciousness and suffocation (HSDB).

Other reported symptoms from 'overexposure' to the chemical include gastrointestinal congestion, low blood pressure, and damage to the liver and kidneys (HSDB).

Genotoxicity

The chemical is classified as hazardous—Category 3 mutagenic substance—with the risk phrase 'Possible risk of irreversible effects' (R68) in the HSIS (Safe Work Australia). The available data support this classification.

The European Food Safety Authority (EFSA) has evaluated the available data on the mode of action for the chemical and concluded that ‘furan-induced carcinogenicity is probably attributable to a genotoxic mechanism. However, chronic toxicity with secondary cell proliferation may indirectly amplify the tumour response’ (EFSA, 2004).

Studies have indicated that a cytotoxic metabolite of the chemical, *cis*-2-butene-1,4-dial (BDA) (see **Toxicokinetics**) plays a role in the genotoxicity of the chemical, due to rapid reaction with protein and nucleic acids. The metabolite BDA was reported to give positive results in the majority of in vitro genotoxicity assays and reacts directly with DNA in target cells, stimulating cellular proliferation and increasing the likelihood of tumour induction (EFSA, 2004; VKM, 2012).

Studies on the chemical reported mixed results in several in vitro assays (IARC, 1995; EFSA, 2004, VKM, 2012; HSDB; REACH):

- negative results in bacterial reverse mutation assays with several strains of *Salmonella typhimurium* at concentrations up to 3333 µg/plate, after preincubation with or without metabolic activation;
- weakly mutagenic in *S. typhimurium* strain TA100, with or without metabolic activation;
- induced chromosomal aberrations and sister chromatid exchanges (SCE) in Chinese hamster ovary (CHO) cells, with or without metabolic activation;
- positive results in the thymidine kinase locus of L5178Y mouse lymphoma cells without metabolic activation;
- negative results in a micronucleus assay with human lymphocytes at concentrations up to 100 mM, with or without metabolic activation; and
- induced DNA double-strand breaks in isolated rat hepatocytes at a concentration of 100 µM.

The negative results obtained from bacterial reverse mutation assays were stated to be a ‘limitation of “conventional” genotoxicity assays'. The in vitro genotoxicity of furan requires the CYP2E1 cytochrome enzyme for activation and inadequate metabolic activation systems (S9) have been applied in these studies. Auto-oxidation of the chemical to DNA reactive substances after a long exposure time may have contributed to positive results (VKM, 2012).

Mixed results were reported in several in vivo studies (NTP, 1996; EFSA, 2004; VKM, 2012). The reports indicated that the chemical:

- did not induce SCE or chromosomal aberrations in a bone marrow micronucleus test in male B6C3F1 mice, administered the chemical intraperitoneally (i.p.) at doses up to 350 mg/kg bw. However, chromosomal aberrations were induced after an extended harvest time at 250 mg/kg bw;
- did not induce SCE, micronuclei, chromosomal aberrations or DNA strand breaks in the bone marrow or peripheral blood of F344 rats orally administered the chemical at doses up to 2 mg/kg bw/day, or in Big Blue rats at doses up to 30 mg/kg bw/day. The authors stated that this was due to limited distribution of BDA to the bone marrow (VKM, 2012);
- formed DNA adducts and induced DNA strand breaks in F344 rat liver at a known carcinogenic dose (2 mg/kg bw);
- induced DNA strand breaks and caused DNA cross-linking in mouse liver at an acute toxicity dose (200 mg/kg bw). At lower doses (2–15 mg/kg bw/day), overexpression of several DNA repair genes was observed in the liver which was considered by the authors as indirect evidence of genotoxicity;
- induced micronuclei and DNA cross-links in mitogen stimulated rat and mice splenocytes. The animals were orally administered the chemical up to 28 days. No DNA damage was observed in non-stimulated splenocytes;
- induced DNA damage leading to strand breaks and DNA-protein crosslinks in the liver of turkey foetuses at low concentrations (2–20 µmol); and
- gave negative results in a sex-linked recessive lethal test in *Drosophila melanogaster*.

The chemical was reported not to induce unscheduled DNA synthesis (UDS) in rat or mouse hepatocytes in vitro or in vivo (IARC, 1995). However, this assay detects only bulky DNA adducts and is unable to detect genotoxicity resulting from misrepair and non-repair and, therefore, is considered unsuitable (EFSA, 2004).

Negative results obtained from the in vivo studies were postulated to be due to being measured in tissues other than the target organ (liver), and inappropriate sampling times used to detect DNA damage. Furthermore, some in vivo studies like the UDS and micronucleus assay in the bone marrow may not be relevant for testing the genotoxicity of this chemical (VKM, 2012).

Carcinogenicity

The chemical is classified as hazardous as a Category 2 carcinogen with the risk phrase ‘May cause cancer’ (T; R45) in the HSIS (Safe Work Australia). The available data support this classification.

The International Agency for Research on Cancer (IARC) has classified the chemical as ‘*possibly carcinogenic to humans*’ (Group 2B), based on inadequate evidence for carcinogenicity in humans, but sufficient evidence for carcinogenicity in animal testing (IARC, 1995).

In a carcinogenicity study (OECD TG 453), F344 rats (n = 70/sex/dose) were administered (gavage) the chemical at doses of 0, 2, 4 or 8 mg/kg bw/day, five days/week for 102 weeks. Interim evaluations were conducted at nine and 15 months. The survival rates for rats at the highest dose were lower than controls due to moribund sacrifices associated with liver and biliary tract neoplasms, and possibly mononuclear cell leukaemia. Hepatocellular adenoma was observed in both sexes at increased incidences, while hepatocellular carcinomas occurred only in males. Cholangiocarcinomas of the liver occurred in all exposed groups and were present in almost all rats during the interim evaluations, reaching 100 % at the highest dose. An increased incidence of mononuclear cell leukaemia was observed in both sexes at 4 or 8 mg/kg bw/day, with the incidence at the highest dose exceeding historical control ranges for gavage studies with corn oil vehicle (NTP, 1993; IARC, 1995; REACH).

In a two-year ‘stop-exposure study’, male F344 rats (n = 50) were administered (gavage) the chemical at 30 mg/kg bw/day for 13 weeks, and then observed for up to two years. In addition to the lesions previously observed, cholangiocarcinomas of the liver occurred at an incidence of 100 % (40/40), and hepatocellular carcinomas at an incidence of 15 % (6/40) in male rats that

survived for at least nine months (IARC, 1995). Benign lesions of the bile duct epithelium found following 13 weeks' exposure progressed into cholangiocarcinomas, and progression continued after cessation of exposure (NTP, 1993).

In another carcinogenicity study (OECD TG 451), B6C3F1 mice (n = 50/sex/dose) received doses of the chemical at 0, 8 or 15 mg/kg bw/day, five days/week for 104 weeks. The survival rates for males and high dose females were lower than controls due to moribund sacrifices associated with liver neoplasms. Significant dose-related increased incidences of hepatocellular adenomas and carcinomas occurred in all mice. A significant increase incidence of benign adrenal phaeochromocytoma was observed in low and high-dose males, and in high-dose females (IARC, 1995; NTP, 1996).

In another two-year carcinogenicity study conducted at lower doses, female B6C3F1 mice were administered the chemical at doses of 0, 1, 2, 4 or 8 mg/kg bw/day. A statistically significant increase in the incidence of hepatic cytotoxicity was observed at ≥4 mg/kg bw/day. The chemical did not produce tumours at cytotoxic doses of 1 and 2 mg/kg bw/day (Gill, 2011).

Activated oncogenes were studied in hepatocellular tumours induced in mice by the chemical. The chemical and BDA were observed to directly activate proto-oncogenes, which may contribute to carcinogenic effects (NTP, 1996).

Reproductive and Developmental Toxicity

No data are available.

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- systemic long-term effects from repeated oral exposure, carcinogenicity and mutagenicity;
- systemic acute effects from oral and inhalation exposure; and
- skin irritation.

Public Risk Characterisation

Given the uses identified for the chemical, it is unlikely that the public will be exposed. Hence, the public risk from this chemical is not considered to be unreasonable.

Occupational Risk Characterisation

Given the critical systemic long-term, systemic acute and local health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise oral, dermal and inhalation exposure are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine the appropriate controls.

Based on the available data, the hazard classification in the Hazardous Substances Information System (HSIS) (Safe Work Australia) is considered appropriate.

NICNAS Recommendation

Current risk management measures are considered adequate to protect public and workers' health and safety, provided that all requirements are met under workplace health and safety, and poisons legislation as adopted by the relevant state or territory. No

further assessment is required.

Regulatory Control

Work Health and Safety

The chemical is recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)* Harmful by inhalation (Xn; R20)*	Harmful if swallowed - Cat. 4 (H302) Harmful if inhaled - Cat. 4 (H332)
Irritation / Corrosivity	Irritating to skin (Xi; R38)*	Causes skin irritation - Cat. 2 (H315)
Repeat Dose Toxicity	Harmful: danger of serious damage to health by prolonged exposure if swallowed (Xn; R48/22)*	May cause damage to organs through prolonged or repeated exposure - Cat. 2 (H373)
Genotoxicity	Muta. Cat 3 - Possible risk of irreversible effects (Xn; R68)*	Suspected of causing genetic defects - Cat. 2 (H341)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)*	May cause cancer - Cat. 1B (H350)

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

Advice for industry

Control measures

Control measures to minimise the risk from oral, dermal and inhalation exposure to the chemical should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used. Examples of control measures that could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health;
- minimising manual processes and work tasks through automating processes;

- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemical has not been undertaken as part of this assessment.

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